Vol. 69, No. 6 Established 1884 June 1952

ARCHIVES OF PEDIATRICS

A MONTHLY DEVOTED TO THE

DISEASES OF INFANTS AND CHILDREN

JOHN FITCH LANDON, M.D., Editor

LEADING ARTICLES IN THIS NUMBER

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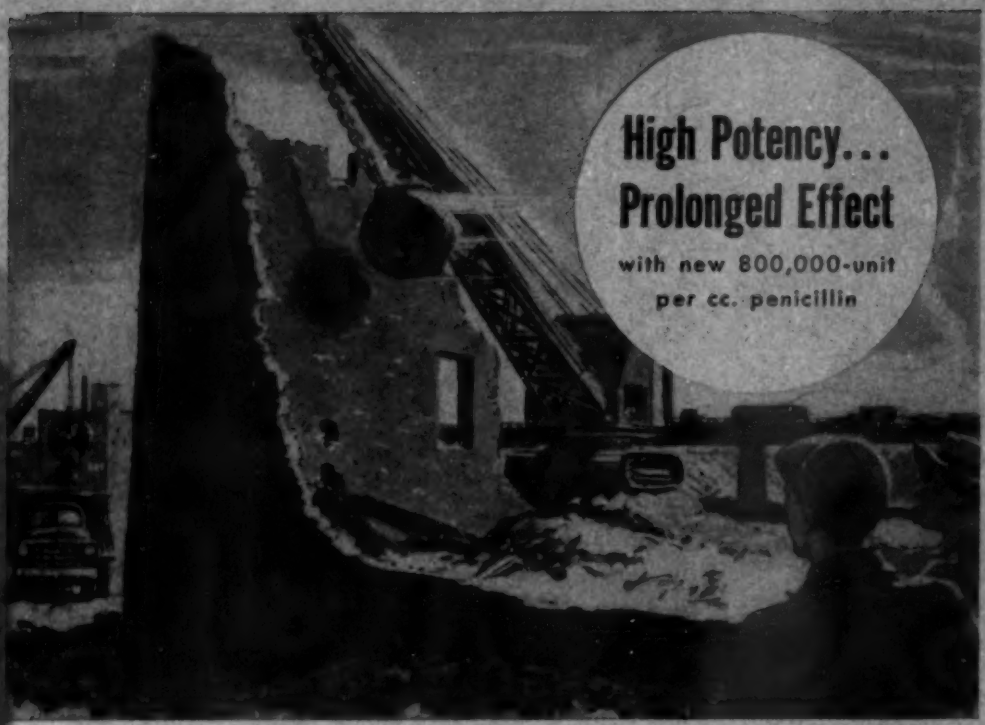
Yearly Subscription $6.00 (Foreign $6.75); Single Copy, $1.00

Entered as second-class matter Feb. 5, 1892, at New York, N. Y., P. O., under the Act of March 3, 1879



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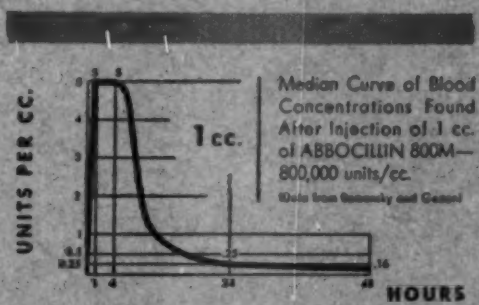

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Editorial Communications address to JOHN F. LANDON, M.D., 120 East 75th Street, New York 21
Business Communications address to E. B. TREAT & Co., INC., 45 East 17th Street, New York 3

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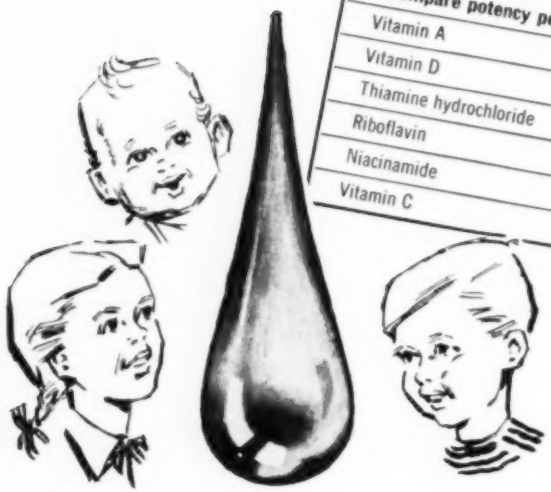

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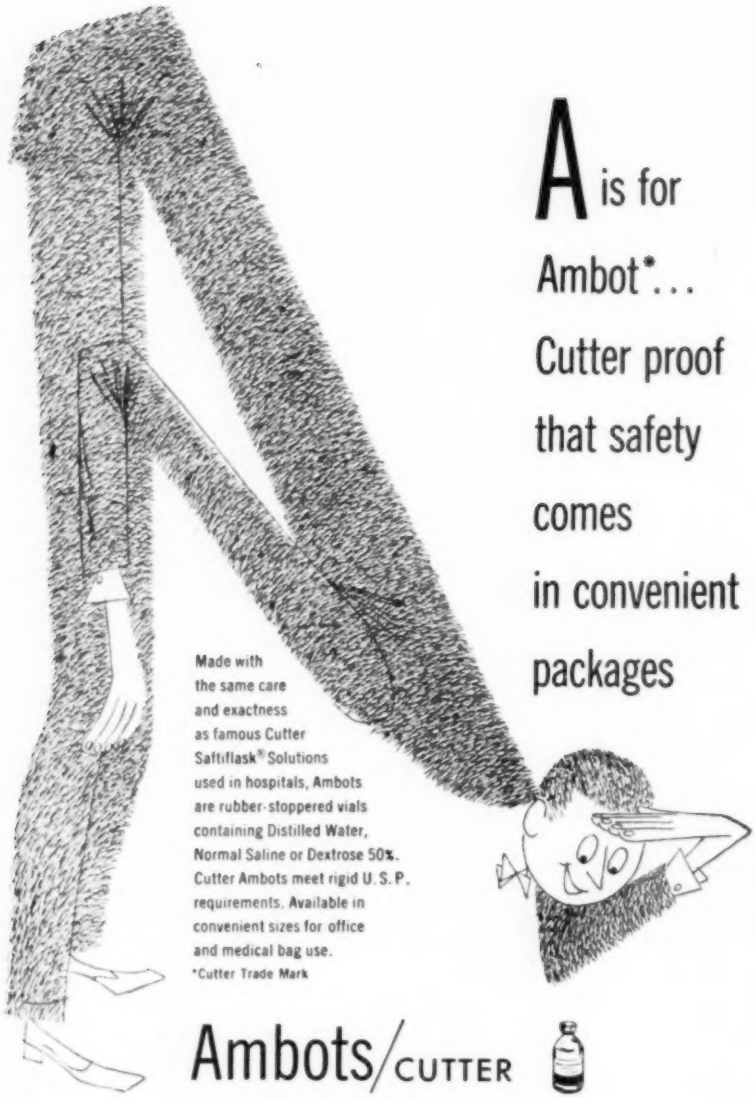

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ARCHIVES OF PEDIATRICS

VOL. 69 JUNE 1952 No. 6

JOHN FITCH LANDON, M.D., Editor

SPONTANEOUS RUPTURE OF THE LENS IN HYDROCEPHALIC EPILEPSY

REPORT OF CASE

GEORGE J. ORLANSKY, M.D.
Palmer, Mass.

A perusal of the literature does not mention the rupture of the crystalline lens in a hydrocephalic epileptic. However, there are reported in the various journals many instances of ruptured crystalline lenses associated with trauma, tumors, infectious, inflammations, toxic states and congenital defects. An evaluation of this pathologic oddity is, therefore, worthy of our consideration.

Anatomical Considerations. The crystalline lens is a biconvex, transparent, circular structure about 11 mm. in diameter and about 3.7 mm. thick at its central point. The center of its anterior surface coincides with the center of the pupil. The lens is enclosed in a structureless, highly elastic capsule. It lies in its capsule behind the pupil of the eye and is kept in place by its suspensory ligament. Histologically the substance of the lens consists of a group of fibers which simulate elongated epithelial cells which fuse with other fibers from the periphery. On section the lens discloses a series of concentric laminae with a nucleus of a high convexity and a less refractive cortex. Embryologically the lens is epithelial and avascular and receives its nourishment from the fluids bathing its surfaces, i. e., the fluid of the vitreous and aqueous humors. The capsule acts as a semi-permeable membrane and thus helps to separate pressure factors.

Senior Physician, Pediatric Service, Monson State Hospital, Palmer, Massachusetts (George J. Orlansky, M.D.)
Photographic Studies by William A. Hunter, M.D., Assistant Superintendent, Monson State Hospital, Palmer, Mass.

Physiological Considerations. The lens serves to refract the rays of light entering the pupil and impinging on its surface so as to bring them to a focus upon the retina and thereby establish the principle of accommodation whereby objects at different distances are clearly seen.

CASE DISCUSSION

This patient was a nine-months-old white male who was admitted to the Monson State Hospital in Palmer, Massachusetts in the fall of 1951 because of a history of convulsions and an enlarged head. On admission he weighed 32½ pounds and was 30 inches in height with head measurements indicating a circumference of 30 inches, transverse arch 22 inches and glabella to inion 23½ inches. There was a postoperative scar measuring 2¾ inches over the anterior head with missing bone over the left portion from the frontal to occiput and many circular lesions on the left side of the head and over the right lateral head. There were many prominent vascular markings. The hair was of thin texture and blonde. The eyes disclosed a rotary nystagmus. The globes were downward. The pupils were equal, but there was no response to light. Vision was absent. Hearing was markedly impaired and probably absent, for there was no response to clapping directly over the face. The heart and lungs were essentially negative. The pulse was 96 per minute, regular and of good quality. The respirations were equal and 14 per minute, the temperature 99.2° F. The nose was of the small, pug type and the mouth disclosed two upper central incisors and one lower. The palate was high and arched, the tongue moist and red. The neck did not disclose any palpable glands. The trachea was in the midline. The neck muscles were restricted because of the inability to move the head, due to its weight and enlargement. The abdomen was slightly protruding with a mild umbilical hernia. The testicles were in situ. Neurologically, the reflexes were intact except for an absent cremasteric. There were no tremors. The tonus was poor. The infant had a whining cry and was totally helpless.

Psychometric examination disclosed an idiot of less than six months' mental age.

The family history disclosed the paternal grandfather to be unstable and alcoholic and the paternal grandmother diabetic. The latter evidently died of mammary cancer at the age of fifty-five.

The maternal grandfather was always inclined to be nervous and developed a spinal paralysis and died at the age of thirty years. The rest of the family history is noncontributory.

Present Illness. The youngster was born following a normal,

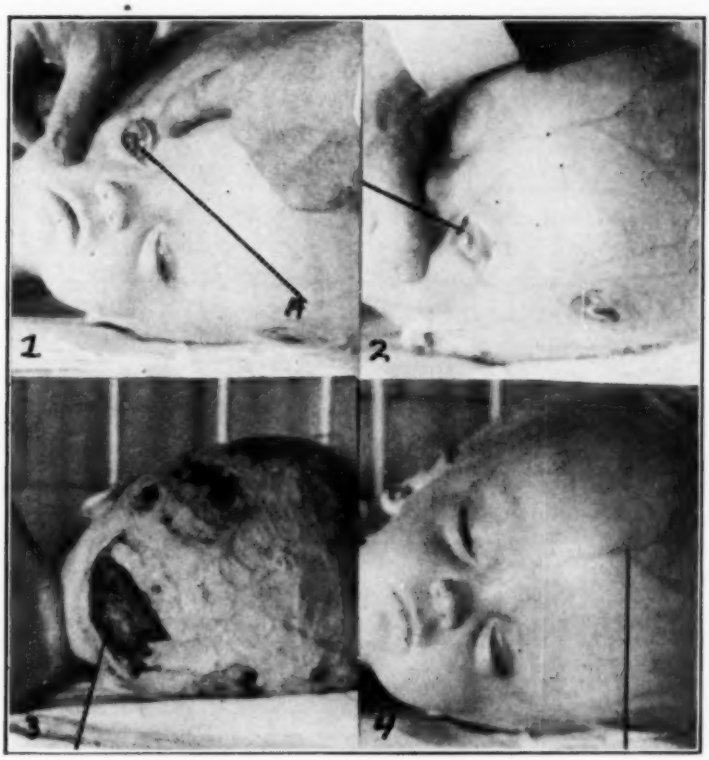

Fig. 1. Point of rupture of lens (A).

Fig. 2. Left eyeball about to rupture. Note broken down tissue at left due to diminished circulation.

Fig. 3. Breakdown of tissues of left head and occiput.

Fig. 4. General hydrocephalic features with prominence of vascular markings.

nine months' pregnancy by a traumatic, breech delivery, weighed 9½ pounds but remained listless for the first few days, presumed to be on the basis of obstetrical anesthesia. Immediately following

delivery he developed signs of increased intracranial pressure and intracranial hemorrhage in addition to a high temperature with generalized sepsis and meningitis which responded to intensive chemotherapy. A left brachial palsy had also been incurred and treated with a brace and improved during the course of the next few weeks. The head, at this time, seemed to be increasing in size, and a twitching of the left eye and left arm was observed.

The laboratory data prior to admission disclosed a hemoglobin of 9.9 gms. with a R.B.C. of 3.4 million and W.B.C. 20,650 (during

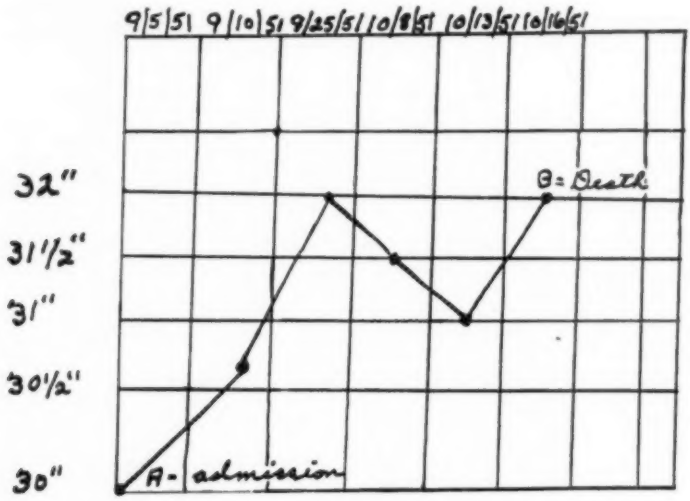

Fig. 5. Head measurements (circumference).

course of developing sepsis). Total proteins on ventricular fluids was 694 mgs.%. Hinton was negative. X-ray study was essentially negative. The findings were those of hydrocephalus, probably on an obstructive basis. Both lateral ventricles were considerably dilated. A sagittal sinus venogram* showed an essentially normal sinus. There appeared to be a depression of the tortula in association with a very small posterior fossa and a depression of the tentorium. Lumbar puncture pressure was 60 with a slow drip and ventricular pressure 150. A conclusion was that the patient

*Work done at Children's Hospital of Boston, Mass.

was probably blocked with insufficient flow at the lumbar puncture site. Ventriculogram showed a marked thinning of the cortex and a dilatation of the lateral and third ventricles. A few days later a repeat lumbar puncture revealed xanthochromic fluid could be obtained before the tap ran dry. The ventricular fluid was lighter in color and the fontanel tense. Forty-eight hours later a sagittal phlebogram was performed which showed no block in the sinus systems of the brain, with a resulting diagnosis of noncommunicating hydrocephalus secondary to a birth injury.

The hospital course was characterized by failing health, increase in circumference of head by two inches, twenty days after admission, with rupture of the right crystalline lens thirty-four days after admission, and varying degrees of multiple cephalic ulcerations and with death resulting forty-one days after admission from terminal bronchopneumonia.

PATHOLOGIC STUDY

Autopsy findings of significance disclosed a white male infant 10 months and 26 days of age weighing 30 pounds and measuring 30 inches in height with the circumference of the head measuring 32 inches. The skin over the head was separated and the suture line from the frontal to the occipital protuberance was separated sufficiently to permit the index finger to enter. The skull was opened easily with a blunt scissor in the anterior posterior diameter. The meninges were excised and there was a gush of fluid, yellow-tinged, which flowed out under marked pressure about one liter in quantity. A collapsed sack of thin membrane remained and what was left of brain tissue was about the size of 3 cms. The bones of the cranium were thinned and translucent. There was considerable thinning of the orbital plate and the shadow of the finger externally could easily be seen internally. The optic nerve was atrophied and there was a small opening in the right eyeball about 12 o'clock through which the crystalline lens came and an area of large scarring involving the anterior portion of the left eye.

THEORETICAL DISCUSSIONS

This patient presented a problem as to the various factors involved in spontaneous rupture of the lens. The chemical changes concerned involved just as to how high the concentration of potassium, calcium, sodium chloride, magnesium, silicates, choleste-

rol and phosphatides, and the total salt concentration may be to produce hardening of the lens. Adams has demonstrated a small but definite oxygen uptake and Goldschmidt has shown an autoxidation system. The mechanical changes involve a series of factors dealing with gradual increased weight; downward displacement of the eyes through the orbit; flattening of the globe with anterior bulging; dislocation of the lens; stretching and tension of the optic nerve and sheath leading to weakness and spontaneous rupture at limbus.

CONCLUSION

A case of spontaneous rupture of the crystalline lens in hydrocephalic epilepsy due to mechanical factors is presented.

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CONGENITAL MALARIA. (Archiv. fur Kinderheilkunde, Stuttgart, 143: 113, 1951). Congenital malaria can be proved only when a mother who has malaria gives birth to a malarial child in a region where there are no Anopheles mosquitoes. Voss presents two cases of malaria that he believes can be classified as congenital and cites reports from the literature. Intrauterine infection of the fetus has been demonstrated repeatedly, but its mechanism has not been explained. The intact placenta acts as a filter, and plasmodial parasites that circulate in the blood cannot enter the tissues. It has been suggested that traumatic lesions of the placenta might be produced by malarial chills and other causes. The hemorrhagic tendency of malaria patients supports this assumption. The author believes that nonviability usually results in cases of intrauterine infection, and that, in infants born alive, infection generally takes place during delivery. Symptoms of malaria usually appear after a latent period of not less than seven days. Congenital malaria behaves like an inoculation malaria in that a single course of quinacrine is adequate for cure, supplementary treatment with pamaquine being unnecessary.—*Journal A.M.A.*

INCONTINENCE OF URINE AND THE NEUROGENIC BLADDER IN CHILDHOOD

With Illustrative Case

Theodore James, M. B., Cape Town
The Duchess of York Hospital for Babies, Manchester, England.

Except for special textbooks like that of Meredith Campbell, and T. Twistington Higgins, William and Nash, both of very recent publication (1951), which discuss the problem and importance of neurogenic vesical dysfunction, or so-called neurogenic bladder it is difficult to find in paediatric literature an adequate exposition of the malady as it occurs in childhood. The standard paediatric textbooks and other books of reference make only brief mention of this disorder but give little or no details. Incontinence of urine and spina bifida have for long had a recognized association, but it is the neurogenic bladder without any obvious pointer to its pathogenesis that has remained much neglected. A few papers treating the disability in childhood have appeared in the special journals but the paediatric journals have shown a noteworthy absence of writings on the subject; it is the purpose of this paper to present the case of the unobtrusive neurogenic bladder. A survey of the literature concerned with this type of vesical dysfunction reveals that it is diagnosed more frequently in adolescents and adults than it is in children and leaves a strong impression that many cases of one or other type of neurogenic bladder pass undected during childhood.

Incontinence of urine in a child is one of the commonest problems which the paediatrician is called upon by a harassed parent to solve. Up to the age of 2 years, day and night incontinence is to be expected, but between 2 and 3 years of age the child should begin to control urination, especially by day. "Accidents" are tolerated by most parents until the child is about 3 years old; if by then little, if any, control has become manifest the parent justifiably becomes perturbed and seeks medical advice. The majority of cases are brought for consultation when they are between 5 and 10 years of age, and many of these have been bed-wetting since birth but have control of micturition during the day. The longer the nocturnal enuresis persists, however, the likelier is diurnal

enuresis to develop. In Campbell's (1951) experience more than half his night-wetters were also day-wetters.

Functional enuresis has been the chief complaint and diagnosis in many cases of neurogenic vesical dysfunction, the true diagnosis being left until much later. In some of these it can be extremely difficult to differentiate between organic neurogenic disease and a purely functional disturbance (Campbell, 1951.)

When the problem of urinary incontinence presents itself, its origin is to be looked for among the following causes, after a careful anamnesis has been taken; and it is as well to bear in mind that neuromuscular uropathy commonly passes unreognized yet is of high incidence in infants and children and that it is always potentially serious and often grave (Campbell, 1951).

Organic Causes. Diabetes mellitus, or insipidus; chronic interstitial nephritis; disease of the brain or spinal cord; profound exhaustion, coma; spinal fusion defects; urethral valves; phimosis; cystitis; impacted calculus; extrophy; abnormal openings of ureters into vagina; persistent urachus; absence of vesical sphincter; partial obstruction from malformation of posterior uretha producing a distended bladder with constant dribbling; trauma of the cord, and *defective innervation of the bladder, producing the neurogenic bladder or neurogenic vesical dysfunction.*

Functional Causes. A sequel to a lowered state of health following nervous disorders, or acute infectious fevers; fright or accident, involving injury, may initiate it; irritation by pin-worms; constipation; septic tonsils and adenoids; carious teeth; indigestion and late meals; long drinks on going to bed; hypersensitivity to the bedding material; a relapse with an infantile behavior pattern; low mentality.

When day and/or night-wetting continues after the age when normal control should be exercised, about 3 years of age, or persists for more than 2 or 3 months after an illness or other disability despite medical management, then a urologic examination is indicated. Campbell (1932) found organic disease in more than half of a series of 249 cases investigated for this reason alone Enuresis has been found as a symptom of almost every kind of uropathy.

Smith and Engel (1932), who regarded the basic etiological factor in neurogenic bladder dysfunction in children to be a de-

fective parasympathetic innervation in most instances, could attribute this to defective spinal development in 8 of 13 cases; in the other 5 there were no fusion defects detectable and 3 of these 5 had retention with ability to void small amounts with great difficulty and the other 2 had frank incontinence without retention. There were 7 of the 13 cases without other evidence of neurotrophic changes, such as saddle anesthesia, trophic ulcers, and varying degress of muscular weakness of the legs.

Mertz and Smith (1930), who were primarily concerned with posterior spinal fusion defects and nerve dysfunction of the urinary tract, collected 26 cases from the literature; 18 of these were children, and added 13 cases, all children, of their own. Case 5 of Brechot, which they cited, a male of 7½ years with enuresis and occasional day incontinence, had no observable anatomic defect, but many of the others had gross fusion defects.

In this connection it is of interest to note the importance or unimportance which different writers attach to occult fusion defects. Higgins et al. are of the opinion that minor fusion defects of the spine are of no significance. Ingraham and Lowrey (1943) state that roughly 25 per cent of normal children have occult defects of the vertebral laminae, which can be shown to persist in normal adults. They reported 65 cases with occult defects which had produced symptoms, and enuresis and/or incontinence was a symptom in 7 of the 65 patients. McLellan (1939), however, gives 50 per cent of routine roentgen studies of the lumbosacral spine as showing a spina bifida occulta without urinary symptoms. This 50 per cent comprises about 90 per cent of spina bifida occulta. Hintze (cit. Mertz and Smith, 1930) dogmatizes that before a "lumbo-sacral fontanelle" can be considered a spina bifida occulta there must be present in the lumbosacral region a tumor, a pigmented area, hypertrichosis, et cetera, and Hoehen (cit. Mertz and Smith) asserts that to diagnose a spina bifida occulta as the cause there must be evidence of neurological changes of the perineum or extremities.

Yet Mertz and Smith have shown that various neurological disturbances have been localized to the bony defect with a frequency sufficient to justify careful consideration of these defects, every one of which adds to the abnormal anatomical relations of the spinal cord and its membranes with the surrounding structures.

In many of the case reports in the literature which were collected by Mertz and Smith, spina bifida occulta was frequently found associated with fibrous bands which compressed the cord and operative removal was often successful, but it should be added that these good results following removal of such bands happended in cases in which the fusion defect was not minimal.

Spina bifida occulta and myelodysplasia have been adduced to explain many cases of enuresis when these 2 conditions have occurred together and it follows that in such cases the enuresis has been looked upon as organic rather than functional. Fuch's (1909) put spina bifida as the cause of enuresis in 50 per cent of cases but Langworthy and Wells (1936) are of the opinion that one-third of all individuals have a spina bifida occulta and yet the defect is etiologically important in neuromuscular disease in not more than 5 per cent of cases.

Mertz (1933) was convinced of a relationship between spina bifida occulta and neuromuscular dysfunction of the urinary tract and he carried out laminectomy in 6 young people of ages ranging from 2½ years to 18 years. Two of these were given definite relief from the urinary disturbance, 2 were slightly improved and in the other 2 there was no appreciable change in symptoms.

He came to a conclusion that laminectomy for the relief of enuresis alone should not be undertaken, although a definite spina bifida occulta exists, before the patient has reached the age of puberty and then if carried out it should be done only after all other measures have failed. He expressed the view that nerve dysfunction is progressive and primarily initative and caused by abnormal tissues filling the osseous defect. This affects the vesical center of the cord to produce vesical evacuation during sleep when there is no psychic control. Such irritability may progress to paralytic change when incontinence or retention will result; the most frequent urinary tract symptom of spina bifida occulta he believed to be enuresis.

NEURO-ANATOMY AND NEUROPHYSIOLOGY

Despite the time-honored clinical association of overt and covert spinal fusion defect and disturbance of micturition, physicians for long were ignorant of the neuro-anatomy and neurophysiology of the normal bladder and pathological aberrations were, therefore, also ill-understood. Only within fairly recent times have studies

established facts necessary for an adequate understanding of the neurogenic bladder as it occurs in adults and children.

It is as well if a brief recapitulation of the neuro-anatomy and neurophysiology be given for a better comprehension of the neurogenic bladder, especially the type in which there is no detectable cause for the dysfunction. This resumé is based upon the work of McLellan (1939), Moore, (1924), Munro (1947), Jacobson (1945), Latchem (1950) and others.

The bladder is connected with the central nervous system by sympathetic fibres from the twelfth dorsal to the third lumbar segments. These fibres pass by way of the superior hypogastric ganglia (the presacral nerve) to each inferior hypogastric ganglion and so to their distribution in the bladder wall. The sympathetic nerves supply the bladder with motor function, and control the tone of the trigone, ureteral orificies and internal sphincter; but, there is also some inhibitory effect on the detrusor. With these nerves run also some vasoconstrictor and visceral afferent filaments.

The parasympathetic nerves arise in the second, third and fourth sacral cord-segments and pass by way of the cauda equina and the pelvic nerves to each hypogastric ganglion before they reach the bladder. The internal pudendal nerves arise from the anterior primary division of the second, third and fourth sacral cord-segments and leave the pelvis to enter Alcock's canal to supply the perineum; and give a semivoluntary innervation to the external sphincter of the bladder, the levator ani and superficial perineal muscles. The motor integrity of this nerve (it contains sensory fibres to the erineum and external genitalia as well) may be determined by the voluntary activity of the external anal sphincter and bulbocavernosas muscle; for the function of the external vesical sphincter is analagous to that of the external anal sphincter.

Completion of the reflex arc is in the second, third and fourth sacral cord-segments and this part of the cord with intact parasympathetic tracts allows reflex micturition. Ascending spinal cord pathways appear to lie in the anterolateral columns and the descending pathways in the pyramidal tracts. Three centers have been postulated for reflex bladder contraction, these being in the lumbar cord, hypogastric plexus and the inferior mesenteric ganglia.

There is still some ignorance of the normal and abnormal neurophysiology of the bladder and some statements which are contradictory; but there is agreement that the presacral nerves or sympathetic nerves have little or nothing to do with micturition.

Barrington's (1914) original formulation for normal micturition was an automatic sequence of 5 reflexes but such a formulation was superseded by that of Munro in whose eyes normal micturition is a reflex act under voluntary control, the reflex centers lying in the sacral segments of the spinal cord. It is a result of reflex contraction of the detrusor muscle followed by reflex relaxation of the external sphincter. The stimulus for detrusor contraction is the result of the bladder filling with stretching of the smooth muscle fibres of the bladder wall, both afferent and efferent arcs of this reflex travel via the parasympathetic nerve. Detrusor contraction causes the internal sphincter to relax reflexly and relaxation of the external sphincter follows making it appear reflexly related to detrusor contraction and/or internal sphincter relaxation. It is apparent that the motivation for bladder evacuation lies within the sacral segments of the spinal cord. Suprasegumental control provides inhibitory influences over the sacral centers and prevents automatic emptying of the bladder with the first impulses of smooth muscle stretch. This vesical distention proceeds further before reaching the level of consciousness or before the beginning of reflex detrusor contraction which allows a still greater storage of urine. Voluntary urination takes place only by a control of inhibitory impulses from the higher cerebral centers, that is, an emptying contraction cannot be *willed* but the inhibitory impulse can be suppressed so permitting the reflex act. Relaxation of the external sphincters occurs as a reflex only (Whitmore and Isales, 1948). The external urinary sphincter serves as a voluntary and as an involuntary muscle; the internal sphincter, although purely involuntary, is not essential for the normal act of micturition (Drexler and Rothfeld, 1950).

The normal function of the bladder is dependent, therefore, upon the integrity of nerve pathways which connect it with the brain. The essential tracts lie in the parasympathetic division of the autonomic nervous system and may be divided into segmental (or spinal) reflex and a conditioning suprasegmental or cerebrospinal reflex. Any interference with these pathways produces

a disturbance of bladder function, that is, the neurogenic bladder (Nesbit and Gordon, 1940).

THE TYPES OF NEUROGENIC BLADDER

The neurogenic bladder has been divided into four types and three have found a wide acceptance among urologists and much of the scientific data for this division derives from the work of Langworthy (1935), Denny-Brown (1933), Lewis (1936) and others.

Type I is the uninhibited bladder which has been further subdivided into the *congenital* and *acquired* subforms. The *congenital* form includes the physiologic infantile bladder with frequency and urgency by day and night. Uncontrolled contraction of the detrusor occurs during filling and this is interpreted as a desire to void (Monteiro). The *acquired* form follows on lesions in the central nervous system. A subtotal destruction of the cortex or spinal tracts (Monteiro) but voluntary control of the external sphincter is possible and micturition may be held back and the contraction of the detrusor made to subside reflexly (Whitmore and Isales, 1948).

Type II is the automatic or reflex bladder. In this type there is involuntary reflex urination—an inability to stop or start it, and a loss of bladder sensation. There is residual urine, and lithiasis and infection are sequelae (Monteiro). Also there is increased tone and a decreased capacity. Although cut off from the higher centers it is functionally the most efficient (Latcham,1950). The reflex centers in the cord are intact as are the afferent and efferent sides of the reflex arc; but there is an interruption between these centers and the higher centers of control. The bladder empties automatically when afferent impulses mount enough to cause a reflex discharge over the efferent arc. The nerve fibres to the external sphincter are also severed and so this type is entirely involuntary. This type also has been subdivided into a normal reflex and a hypertonic reflex form. The first kind may or may not have residual urine, voids at widely different intervals and in varying amounts. It may be quite precipitate, whereas the hypertonic kind voids small amounts at frequent intervals. Residual urine is little or absent (Whitmore and Isales) and the vesical neck is normal in this type (Jacobson, 1945).

Type III is the autonomic bladder where there is an interruption of the reflex arc, that is, an interference with the extrinsic nerve supply of the bladder whose function is managed by the intramural nerves or pelvic plexus, or both. Marked trabeculation occurs, normal vesical sensibility is absent and voluntary micturition and the reflex are abolished. There is always residual urine and there may develop dilatation of ureters, pelves, and obstruction at the vesico-ureteral junctions which produces detrusor hypertrophy (Monteiro). This type involves the conus or cauda equina with interruption of the motor and sensory sides of the reflex arc. There is increased tone and decreased capacity. There may be feeble, poorly coordinated contraction. It is a less efficient type (Latchem). There may be a dribbling incontinence from the high intravesical presssure and the internal sphincter may be spastic (McLellan, 1939). Clinically there may be incomplete retention with overflow, incontinence or feeble contractions producing the evacuation of only a few millilitres of urine at a time. Voiding is aided by abdominal straining, and bladder capacity is about normal (Whitmore and Isales).

Type IV. In this type there is interruption of only the sensory limb of the reflex arc and this produces a great bladder capacity with an overflow incontinence. With the absence of sensation there is a large volume of residual urine (Nesbit and Gordon). It is the type which is seen in spinal shock (Whitmore and Isales) and the low intravesical pressure and absent sensativeness is accompanied by absence of urge to urinate. The bladder is huge and straining helps voiding. The bladder developes a typically characteristic funnel-shaped outlet, the internal sphincter being relaxed. There is much residual urine (McLellan, 1939). The atony of the vesical musculature is secondary to prolonged distention which produces a false incontinence (Monteiro). Damage to the conus or cauda equina or posterior roots or pelvic nerves may result in little or no reflex activity (Latchem). This is the so-called atonic bladder.

These types are not wholly comprehensive for the neurogenic bladder as it occurs at any age, but they are the best attempt at a classification up to the present time. Paediatric urologists may regard them as of little practical, that is therapeutic importance, but a knowledge of these types is essential to an understanding of

the problem involved when a patient with urinary disturbances seeks advice.

The clinician will see only 2 manifestations of these 4 types. The one is a reflexly emptying bladder without voluntary contraction, the sequel to supranuclear disease, and the other shows an absence of voluntary or reflex activity, emptying is done by increasing the abdominal pressure. This clinical type is the result of nuclear or infranuclear disease.

All 4 neuropathic types occur in children but the commonest one met with in paediatric practice is the autonomic type involving the conus and/or cauda equina. These myelodysplasias, deformities of the cord or cauda equina itself, usually accompany bony defect of the vertebral column which is demonstrable by roetgenography. With the bladder disturbance there may be neurologic change in perineum and lower extremities which may be deformed. The only presenting symptoms of this neurologic defect may be incontinence, although we are becoming more and more aware that pyuria may be a complication of neurogenic vesical dysfunction (Latchem). The young patient who gives a history of chills, fever, and pyuria which fails to clear up after modern antibiotic therapy, or which recurs after only an apparent cure by this means, even though neurologic findings be absent, may well have a neurogenic bladder disturbance and a urologic examination might show trabeculation, residual urine and changes in the vesical neck.

In his discussion on enuresis McLellan (1939) regards a persistent enuresis which appears to be due to a *failure of developement* or normal supranuclear inhibition over the infantile bladder activity as neurogenic, such in effect are instances of neurogenic bladders. Further developement of suprasegmental inhibition is unlikely at the age when such cases are usually encountered and the *infantile* type of bladder becomes permanent. McLellan (1939) found this type of infantile neurologic bladder in a series of cases aged 20-30 years and in these there were no neurologic findings and spina bifida fusion defects were not a feature in any. A significant development is the progression through an overflow incontinence during the period retention to a state of relatively complete breakdown of neurophysiology which ends in frank incontinence (McLellan).

A lax anal sphincter, paralysis of the legs, dysuria, dribbling

and painful retention and even painful defecation or urinary infection may be associated (Mertz 1933) and disturbances of gait, deformities and muscle weakness (Ingraham and Lowrey) accompany neural abnormalities in spinal fusion defects. The urinary disturbances which may be present with fusion defects include enuresis, urgency, occasional day incontinence, or day frequency, perhaps dating from birth. An enlarged bladder with residual urine, a chronically inflamed bladder mucosa, many fine trabeculations and reflux up the ureters may also be in evidence (Mertz and Smith, 1930). Perhaps retention with ability to void small volumes with great difficulty may be a symptom, but enuresis or diurnal frequency or dribbling may be absent. Fecal incontinence (encopresis), complete anesthesia of the buttocks, perineum and posterior half of the external genitalia (saddle anaesthesia) may be neurologic findings and trophic ulcers are neurotrophic changes commonly found with spina bifida.

ILLUSTRATIVE CASE REPORT

J. M. (No. 26854) was 4½ years old when she was brought for the first time to the medical out-patient department of the Duchess of York Hospital for Babies, Manchester, for the chief complaint of persistent urinary incontinence.

Past History. When 19 months of age J. M. had been hospitalized for alleged vitamin deficiency. At that time she was a fat and flabby child with a prominent abdomen. There was some accompanying lumbar lordosis and a generalized hypotonia. There was some beading of the ribs and expanded epiphyses of the wrists. X-ray examination of the wrists showed some rarefaction of the bones and also suggested a mild scorbutus and healed rickets. Urinalysis at this time also revealed the presence of pus cells. Her weight was 10 Kg. As the child grew her infantile enuretic behavior pattern continued without intermission. She developed sufficient control to void voluntarily about four times a day, but between these voluntary voidings a dribbling of urine continued, otherwise she was of clean habits but a weakness of anal control manifested when she became excited. The mother had had two previous pregnancies which had ended in stillbirths, cause unknown.

Physical Examination. J. M. weighed 16.5 Kg. and was a healthy-looking girl of stated age, but of neglected appearance

Her skin was dry. Her head-circumference measured 49.5 cm and was of normal shape. There was a mild follicular sepsis of her tonsils. The abdomen moved freely with respiration and was not resistant to palpation, but it was scaphoid in its upper half and a slightly bulging fullness was evident in its lower half. The colon was loaded, but the hypogastric swelling was determined to be the bladder reaching to the umbilicus. Palpation of the

Fig. 1. Cystogram of a neurogenic bladder showing dilatation, trabeculation and diverticulation of the bladder wall.

bladder was not painful and moderate pressure upon it expelled urine *per urethram* without pain. A spina bifida was not present clinically but there was a slight scoliosis. The genitalia were those of a normal immature female, there being only a slight vulval erythema. No abnormally placed ureteral orifice was detectable and the urethral and vaginal orifices were normal. Digital exami-

nation per rectum disclosed a lax anal sphincter. All the deep tendon reflexes were present and normal and the superficial reflexes also.

Laboratory Data and Special Examinations. The hemoglobin was 80 per cent, erythrocytes 4.4 million, the leukocytes 9,000 with a differential count of polymorphonuclears 40 per cent, lymphocytes 58 per cent, eosinophils 1 per cent and monocytes 1 per cent. The blood urea was 25 mg. per cent, the erythrocyte sedimentation rate 3 mm. in the hour. A catheter specimen of urine was acid in reaction, had a faint trace of albumin; acetone and sugar were absent. There were many bacteria in the centrifuged deposit and leukocytes were 4 per cent high-power field. There were a few epithelial fragments. A culture grew a coliform organism on three occasions. An intravenous urogram clearly demonstrated the normality of the renal pelves and ureters. After an attempt at voluntary voiding, aided by abdominal compression, there remained 150 ml. of residual urine. Cystography showed a large bladder with trabeculation and small diverticula, and an irregular outline with a lax vesical neck (see Fig. 1).

Cystoscopy proved the ureterovesical orifices to be normal. Fine trabeculation was marked and there was some inflammatory swelling about the bladder neck. An x-ray examination of the spine revealed a minimal spina bifida occulta of the fifth lumbar vertebra.

COMMENT

The features of this case place it in the fourth or atonic group of neurogenic bladders which postulates an interruption of the sensory part of the reflex arc, the consequence of some lesion or other involving the posterior sacral ganglia and interfering with the normal transmission of sensory impulses from the bladder to the cord, the atony being secondary to the prolonged distention which follows the absent sensibility which precludes a desire to urinate. The atony of the detrusor appears to have been a factor encouraging a progressive increase of bladder capacity although J. M. had learned with some aptitude voluntarily to strain at intervals and so to void some of the bladder content but at her young age the absence of any urgency or desire to micturate readily encouraged the development of a false incontinence.

The information that infected urine had been discovered in J. M. when she was 19 months of age gives good reason to believe that the coliform bacillary invasion which was present was of long duration; but in spite of this and the distention with trabeculation of the vesical wall dilatation of the upper reaches of the urinary tract had not as yet taken place, but there was the prospect that in time the ureterovesical valves would become deficient and so allow the complication.

Not many urologists would attribute the neural disability in this case to the very mild fusion defect of the fifth lumbar vertebra only, for the x-ray examination did not show up any defect in the sacral part of the spine. However, some few urologists might point to cases in their experience and to others which have appeared in the literature and remark that there is often little or no relationship between the site per se and extent of the spinal defect, and the neurological findings, although sacral defects are very much more constantly associated with and responsible for vesical dysfunction.

J. M., therefore, was a girl who by her mother's observation had never been "dry" at any time, but because the child herself made no complaint, had been allowed to continue in this way until she was 4½ years old before parental concern was seriously aroused.

SUMMARY

An attempt is made to re-state the significance of urinary incontinence in childhood and its possible connection with neurogenic vesical dysfunction.

A brief account is given of the etiological factors which various authors have adduced to explain the neurogenic bladder.

A summarized version of the neuro-anatomy and neurophysiology of the bladder is submitted as an aid to the better comprehension of the different types of neurogenic bladder which are subsequently described.

An illustrative case report is presented.

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STREPTOMYCIN IN TUBERCULOUS MENINGITIS. (Lancet, London, *2*: 515, Sept. 22, 1951). This paper describes the results of streptomycin treatment in 82 cases of tuberculous meningitis observed by the authors between August 1947 and June 1950. Thirty-six of these children survived and were followed up by physical, radiological, audiometric, and psychometric methods. Twenty-two of them had no residual physical or mental defect. Three children are of exceptional intelligence. Four had some physical defect but were mentally normal. Eight were retarded, with or without physical defects. Two are still under treatment and apparently have no mental defect. The survival rate in early cases was 73.7 per cent and in advanced cases 34.6 per cent. Of 27survivors in the early or intermediate stage, 25 retained normal intelligence 6 of 9 survivors who were in the advanced stage became mentally retarded. All the retarded children were under the age of 3 years on admission. There was no significant difference in the survival rate of patients with meningitis with and without clinical evidence of miliary tuberculosis. Of the latter group, meningitis developed in 10 while they were undergoing treatment for miliary tuberculosis, and 7 survived. Special emphasis is laid on early diagnosis and on the importance of concentration of cases in special centers with the necessary laboratory facilities.—*Journal A.M.A.*

COEXISTING COCCIDIOIDOMYCOSIS AND TUBERCULOSIS IN CHILDREN*

Report of Three Cases

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The purpose of this article is to report coexisting infections of coccidioidomycosis and tuberculosis in each of three patients. The cases include one with primary pulmonary tuberculosis and primary pulmonary coccidioidomycosis, one with disseminated coccidioidomycosis and primary pulmonary tuberculosis, and one with primary pulmonary tuberculosis and tuberculosis of the cervical lymph glands together with primary pulmonary coccidioidomycosis.

Case 1. (No. 247138) This 6-year-old Mexican female was first seen in January 1951 with the complaint of cough and weakness. A chest x-ray revealed a lesion in the left upper lung field. Mantoux 1:1000 and coccidioidin 1:100 were positive. An uncle had died of tuberculosis, but had not been in contact with this child. Review of systems: No pathological symptoms. Developmental history: Normal. Immunizations: Diphtheria, pertussis and tetanus, three injections, and smallpox vaccination. Past illnesses: Measles several years prior to present illness.

Physical Examination. Eyes, ears, nose and throat: No pathological findings. Chest: Clear to percussion and auscultation. Heart: No murmurs, arrythmia noted, no enlargement. Abdomen: No masses palpable; abdomen soft. Genitalia: Normal. Extremities: No pathological findings.

Course. The patient was admitted to the hospital July 2, 1951 with positive gastric cultures for tuberculosis and a positive complement fixation test for coccidioidomycosis within the range for primary pulmonary lesions.

Laboratory Data:		SMEARS	CULTURES
Sputums	4-11-51	negative	positive for Tbc
	4-17-51	negative	positive for Tbc
	4-25-51	negative	negative
	7-21-51	negative	negative

*From the Department of Pediatrics, Kern General Hospital, Bakersfield, Calif.

7-22-51.....negative.....negative
7-23-51.....negative.....negative

Complement Fixation for Coccidioidomycosis		1:2	1:4	1:8	1:16	1:32
4-25-51.....	Positive.....	4	4	0	0	0
	(Primary pulmonary range)					
7-6-51.....	Positive.....	3	1	0	0	0
3-5-52.....	Negative....	0	0	0	0	0

Urines—negative for pathology

	R.B.C.	HGB.	W.B.C.	LYM.	MON.	SEG.	EOS.	SED.	HCT.
7-3-51..	4.05	13.3 Gm.	13,500	24	4	65	6	45	44
5-5-52..		12.3 Gm.	10,400	27	9	58	6	37	42

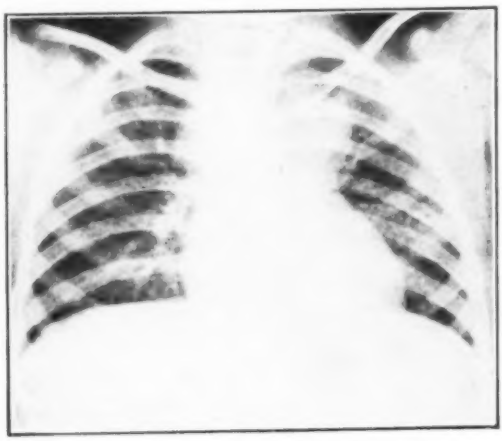

Fig. 1. Linear streaking involving left apex and subclavicular region. Superior mediastinal swelling on right suggestive of a large lymph node.

X-rays. June 7, 1951: Chest—infiltration in the left apex and third anterior interspace (Fig. 1). July 30, 1951: Chest—linear streaking involving left apex and subclavicular region. On the right there is a superior mediastinal swelling suspicious of a large lymph node involvement. August 22, 1951: Chest—considerable clearing of the infiltration in the left apex. March 5, 1952: Chest—lung fields are clear.

Treatment. Streptomycin one gram daily and Promizole 0.5 gram b.i.d. were given from July 2, 1951 until September 7, 1951. As a rule Streptomycin is not given in primary pulmonary tuber-

culosis. In view of active coexisting coccidioidomycosis, Streptomycin was given in order to give the child every possible chance for a cure.

Case 2 (No. 219936). This 3-year-old Mexican was first admitted in January 1950 for influenzal meningitis and otitis media. He was discharged cured February 7, 1950. 2nd admission: The patient was readmitted July 14, 1951, with a croupy cough and vomiting, which had occurred intermittently for one month. The patient had had convulsions during the two days before admission. The father was a known case of pulmonary tuberculosis.

Physical Examination. Eyes, ears, nose and throat: Tonsillopharyngitis. Chest: Râles were heard bilaterally throughout all lung fields. Heart: No murmur, arrythmia or enlargement noted. Abdomen: Liver barely palpable. Diagnosis: Laryngotracheobronchitis and pulmonary tuberculosis.

Laboratory Data. Skin tests with coccidioidin 1:100 and Mantoux 1:1000 were positive. Spinal tap July 16, 1951, revealed four cells.

	R.B.C.	HGB.	W.B.C.	LYM.	MON.	EOS.	SEGS.	STABS.	SED.	HCT.
7-16-51...	3.54	8.4	15,800	18	1	8	67	6		
10-29-51..	4.15	9.6	15,000	35	2	5	52		24	39
2-25-52...	4.42	11.8	9,300	29	7	0	64			

Gastrics	Cultures for Tuberculosis
7-17-51	negative
7-12-51	positive
7-16-51	positive
10-14-15-31-51	positive
1-16-17-18-52	negative

Spinal. October 10, 1951: Cultures and guinea pig negative for tuberculosis.

N.P.N.-31 (July 16, 1951): Urines—negative.

Coccidioidomycosis Complement Fixation Tests:	1:2	1:4	1:8	1:16	1:32	1:64	1:128
10-21-51—disseminated	3	3	3	3	3	0	0
11-2-51—disseminated	3	3	3	3	3	0	0
12-28-51—disseminated	2	2	2	2	2	0	0
2-1-52—disseminated	3	3	3	2	1	0	0

Course. The patient developed measles on August 2, 1951. In November 1951 the patient developed a coccidioidomycosis abscess on the forehead. X-rays of the skull showed two osteolytic lesions

of the skull. Coccidioidomycosis spherules were recovered from the abscess on the forehead, and the mycelian form was cultured. Chest x-rays showed a progressive infiltration in the right apex and infiltration along the left cardiac border (Fig. 2). A skeletal survey showed lesions in the skull, a cavity in the right upper and middle lobe on December 28, 1951. The lesions in the skull showed 25 per cent regression by January 1952. The patient developed varicella in January 1952.

Treatment. Streptomycin, one gram daily, was given for the tuberculosis. The patient was given several blood transfusions. The lesions on the skull were treated with twenty per cent aqueous

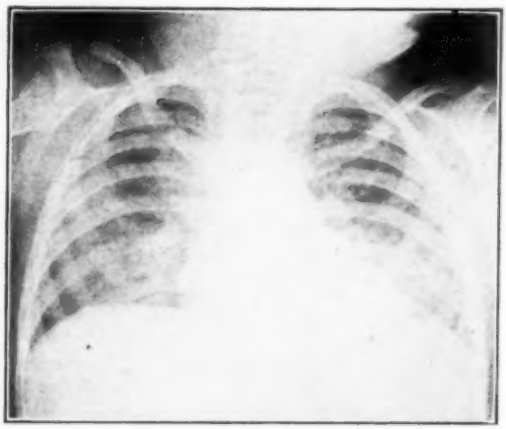

Fig. 2. Infiltration right apex. Note heavy upper hilar markings.

sodium caprylate locally which stopped active purulent drainage and was followed by granulation. Because of the disseminated coccidioidomycosis the patient was started on Stilbamidine[4,5] which has been used successfully in blastomycosis and has been found to be effective against coccidioidomycosis in vitro.[4] This drug was used intravenously for 30 days, starting with 30 mgm. daily and working up to 150 mgm. daily during the last 18 days. On February 29, 1952, the patient developed convulsions, coma, irregular respirations, and a decerebrate type of rigidity. Spinal tap showed no cells. The patient expired on February 29, 1952. An autopsy revealed acute yellow atrophy of the liver, fatty infiltration in the

brain, and a fatty infiltration of kidney tissue. The right upper lung revealed a coccidioidomycosis granuloma; the skull contained two coccidioidomycosis granulomas. Death was attributed to toxic effects from Stilbamidine, effects not mentioned in any other reports on this drug in the literature.

Stilbamidine has been used successfully in blastomycosis and in myelomas. We feel that the drug when used should be evaluated with frequent liver function tests as we have used it in three other cases without untoward effects.

Case 3 (No. 219851). This 9-year-old colored female developed cervical adenopathy in June 1951. An aunt living with her was discovered to have tuberculosis. The glands in the patient's neck

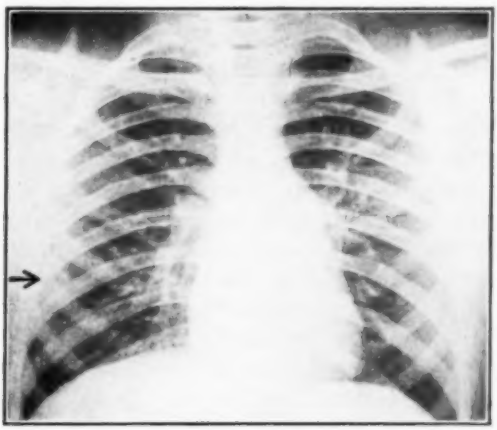

Fig. 3. Increased markings bilaterally and a rounded density suggestive of coccidioidoma or tuberculoma.

were large and confluent and fluctuant. Mantoux 1:1000 and coccidioidin 1:100 in August 1951 were positive. The patient was afebrile and asymptomatic and was admitted with a diagnosis of pulmonary tuberculosis, scrofula, and primary pulmonary coccidioidomycosis.

Laboratory Data. Complement fixation test for coccidioidomycosis on August 22, 1951, was positive in the primary pulmonary range (2-1-0-0-0-0). H.H.B. 13.5 gm. W.B.C. 8,800, lym. 36, mon. 1, eos. 3, bas. 1, segs. 59, sed. rate 38, Hct. 44. Urine nega-

tive. Gastric washing cultures for tuberculosis on August 15, 1951, and September 25, 1951, were positive. Gastric cultures for tuberculosis on December 20, 21 and 22, 1951 were negative. Smears from the cervical glands were negative for tuberculosis but cultures and guinea pigs were positive for tuberculosis (December 31, 1951 and August 15, 1951).

X-ray of chest showed increased markings bilaterally and a rounded density in the right lower lung field in August 1951 (Fig. 3). In February 1952 the x-ray showed a slight clearing of the rounded density at the right base.

Course. The scrofula lesions healed by April 10, 1952.

Treatment. No therapy was given besides bedrest and vitamins.

DISCUSSION

In an endemic area of coccidioidomycosis, co-existing diseases are not uncommon, such as mentioned in this paper. The diagnosis is simple because of the armamentarium of skin tests, cultures and complement fixation tests. It is more prevalent in the dark-skinned and Latin races. The severity of the coccidioidal diseases are evaluated by repeated complement fixation tests, sedimentation rates and chest x-rays. The tuberculosis aspect we feel should have the benefit of Streptomycin if the patient does not appear to be improving by serial x-rays after 4 to 6 weeks.

The autopsy report (Case 2) revealed no active tuberculous lesions in the lungs; the disseminated coccidioidomycosis was still active, however.

CONCLUSION

Three cases of coccidioidomycosis with tuberculosis are reviewed with a discussion of management. These cases are found infrequently but do exist and carry a moderately good prognosis if the coccidioidomycosis is not severely disseminated.

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COARCTATION OF THE AORTA AND PATENT DUCTUS ARTERIOSUS*

Report of Case

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A 5-year-old negro male was admitted to the pediatric service with the chief complaint of easy fatigue on exertion, several severe episodes of epistaxis within the past several months prior to admission, and the discovery of a heart murmur by the school physician during a routine preschool examination. The child was referred to another clinic and then to the New York Medical College, Flower and Fifth Avenue Hospitals' pediatric service for further study.

The child's past history was essentially noncontributory. Growth and development had been normal as far as could be ascertained. Except for measles and an occasional upper respiratory infection, the child had been in apparent good health.

The family history was noncontributory. A review of the systems only reiterated the chief complaints.

Physical Examination revealed an alert, bright-looking 5-year-old boy in no apparent distress. The height was 43¼ inches, the weight 36 pounds. Pulse 120 per minute; respirations 22 per minute. The blood pressure readings were as follows: Right arm 165/85, left arm 150/100; right leg 90/60, left leg 90/60. Eye, ear, nose, and throat were within normal limits. Opthalmoscopic examination revealed normal fundi. The lungs were clear to percussion and auscultation. There were no dilated veins on the neck or chest. A fairly marked pulstation was apparent in the suprasternal area. Examination of the heart revealed a rate of 120 per minute with apparent regular rhythm. The point of maximum impulse was in the fifth left intercostal space at the midclavicular line. Several examiners believed that the left cardiac border was somewhat enlarged to percussion. A loud, harsh, low pitched systolic murmur was heard over the entire precordium. It was also heard in the upper portion of the back. The murmur was loudest in the second and third interspaces, just to the left of the

*Submitted as partial fulfillment of the requirements of the course in Senior Pediatrics at the New York Medical College, Flower and Fifth Avenue Hospitals, New York.

strenum. The murmur was transmitted into the neck and left supraclavicular region. A soft diastolic murmur was reported by one examiner to be heard in the second left interspace. A marked thrill was palpable in the second left interspace and also in the suprasternal region. Abdominal examination was within normal limits. The liver and spleen were not palpable. Examination of the lower extremities revealed absence of arterial pulses in the femoral, popliteal and dorsalic pedis arteries.

Neurological Examination was normal. The hemoglobin was 12.5 grams. The color index 0.8; R.B.C. 4,600,000; W.B.C. 7,400; the differential count was 62 per cent polymorphonuclears, lymphocytes 33 per cent, eosinophils 3 per cent and mononuclears 2 per cent. The sedimentation rate was 20 mm. in one hour. The venous pressure in the right arm was 200 mm. The circulation time from arm to tongue was 15 seconds; the arm to lung time was 4 seconds. X-ray examination of the chest revealed a cardiothoracic ratio just beyond normal limits. The lungs were essentially negative. Angiocardiography revealed a definite narrowing of the aorta just below the arch and great vessels, showing the presence of coarctation of the aorta. A patent ductus arteriosus was also present in the angiocardiogram, but it was not reported at the time of examination of the films. The electrocardiograms revealed slight right axis deviation. They were otherwise within normal limits.

A diagnosis of coarctation of the aorta was made. After consultation with the thoracic surgery service, it was decided to subject the child to surgical repair of the coarctation. Surgery had to be postponed, however, due to the occurrence of a typical case of infectious mononucleosis in the child. The child was ill with low grade fever, generalized lymphadenopathy, splenomegaly, and hepatomegaly for approximately three weeks during which time he exhibited the classical laboratory findings of infectious mononucleosis.

Upon recovering from this complicating episode, the child was discharged to a convalescent home. He was readmitted to the hospital eight weeks later for elective surgery. Physical examination was the same as upon the previous admission. A liver work-up was done in order to determine whether there was any residual liver damage. The cephalin flocculation test was negative. The

serum proteins were within normal limits as was the alkaline phosphatase determination.

The child was prepared for surgery, and three weeks after admission a thoracotomy was performed which revealed a large patent ductus arteriosis equal in caliber to the aortic arch. A marked constriction of the aorta was present just distal to the point of entrance of the ductus arteriosus. The surgical repair consisted of ligation of the ductus arteriosus and resection and primary anastomosis of the aorta at the point of constriction. The immediate postoperative recovery was uneventful. The child was kept in an oxygen tent in view of the pneumothorax produced by thoracotomy. Immediately postoperatively, pulsations appeared in the dorsalis pedis, popliteal, and femoral arteries of both extremities. The blood pressure readings in the upper and lower extremities were as follows: Right arm 140/76, right leg 98/76.

Postoperative roentgenograms of the chest revealed patchy atalectasis in the left lung, mild clouding of the right lung, and a pleural effusion in the lower two-thirds of the left pleural cavity. These findings resolved in about seven days, with only a few small patchy densities remaining in the left lung field which were suggestive of pleural reaction. One week postoperatively, the bloodpressure readings were as follows: Right arm 150/70, right leg 105/84.

Examination of the chest revealed the continued presence of the systolic murmur which was heard preoperatively; however, the diastolic murmur had apparently disappeared. It was felt at this time that the intensity of the systolic murmur in the second left interspace had diminished.

Two weeks postoperatively, the blood pressure in the lower extremities was of the order of 110/86. The operative wound was well healed, and the child was found to be alert, happy, and running about.

It should be noted that reexamination of the preoperative angiocardiograms was made, and that the patent ductus arteriosus could be visualized.

SUMMARY

A case of combined coarctation of the aorta and patent ductus arteriosus is presented. The coarctation was of the "adult type." Successful surgical repair of both lesions was performed, and was

followed by increased blood supply to the lower half of the body.

The decision to surgically repair a coarctation of the aorta was based upon the presence of symptomatology and cardiac enlargement; not merely upon the presence of the defect itself.

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PSYCHIC INCONTINENCE OF FECES (ENCOPRESIS). (Revista Española de Pediatria, Zaragoza, *7*:15, Jan.-Feb. 1951). Six children with psychic incontinence of the feces (encopresis) are reported on. The disease is evident in preschool and school-age children. It is most frequent among neuropaths who come from homes in which the moral, economic, and educative conditions are not favorable. Pathogenically, two mechanisms for the production of encopresis are accepted: (1) an increase in the rapidity and intensity of the defecation reflex, making voluntary control difficult, and (2) a decrease or lack of cerebral inhibition. A third factor is also evident in some cases, namely, hypotonia of the anal sphincter. The author concludes that the prognosis in encopresis is favorable. In the aforementioned cases, the treatment, which consisted of psychotherapy, produced good results in all cases.—*Journal A.M.A.*

PENICILLIN IN GONORRHEAL VULVOVAGINITIS. (Wiener klinische Wochenschrift, Vienna, 63: 261, April 6, 1951). Twelve female infants with gonorrheal vulvovaginitis were treated with penicillin. Fifty thousand units of the antibiotic were injected every three hours, with a total dose of 500,000 units. Local treatment was not given. The author advises against hormone therapy as an unphysiological procedure in the infant. Untoward reactions to penicillin were not observed. All the infants made a complete recovery. They remained in the hospital for an average period of one month. Cultures and microscopic follow-up studies were done for six months. Despite the unfavorable reports by other workers, penicillin therapy is considered the method of choice for gonorrheal vulvovaginitis in infants, provided that the adequate total dose of 500,000 units is administered. Combined treatment with estrogens or sulfonamide compounds is not necessary.—*Journal A.M.A.*

PEDIATRICS AT THE TURN OF THE CENTURY

From time to time the Archives, which was the first Children's Journal in the English language, will reprint contributions by the pioneers of the specialty forty to fifty years ago. It is believed that our readers will be interested in reviewing such early pediatric thought.

DIPHTHERIA IN EARLY LIFE*

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HIDDEN NASAL DIPHTHERIA WITH SEVERE SYSTEMIC INFECTION

During the past ten years the presence of diphtheria bacilli in the nasal secretions of young children suffering from "catarrhal rhinitis" has been reported repeatedly. Among seventy-five children with acute rhinitis, Stoos found the diphtheria (or the pseudodiphtheria) bacillus in 72 cases. As early as 1894 Heubner described cases of "larvirter" diphtheria. Others reported on their findings of this bacterial variety in the upper air passages of healthy children, especially in public institutions (Eric Müller, G. Raser, Chatin, Lessieur).

In the first case reported here, it will be shown that in hidden diphtheria only the positive finding of Löffler bacilli can be of diagnostic value.

J. C., female, aged twenty-three months, was attacked with acute rhinitis in February 1902, which, excepting pallor and mouth-breathing, caused no annoyance to the strong child during the first four days, not even anorexia or unwillingness to play. Antiseptic spraying of the nares had been done very frequently. On the fifth day of illness (March 1) the child appeared cross and tired, and the father (a medical man) found a rectal temperature of 105° F. The family physician, who was then called in, found marked rhinitis, excoriations at the nasal entrances and a small amount of albumin in the urine. The secretion from the nose appeared clear and watery and contained no pus or blood.

*Read at the meeting of the German Medical Society, January 4, 1904.
At the time of writing this paper the author was Professor of Pediatrics, New York Polyclinic, New York City.
Reprinted from ARCHIVES OF PEDIATRICS, 22: 116-123, February 1905.

Following the correct idea that, in spite of the absence of pseudomembranes on the visible nasal mucosa, true diphtheria might be present in this case, the family physician sent some of the secretion to the bacteriological laboratory of the Health Department. No Löffler bacilli were found.

As the temperature of the child remained high (between 103.5° and 105° F.) during the next few days, otitis media was thought of, although no complaints of pain had been made by the patient. An otologist found both drums somewhat bulging, and performed bilateral paracentesis, but very little serum escaped and the temperature remained the same.

When I met the three physicians at the bedside of this patient on the eighth day of illness, I found a strong, well nourished child, which showed no traces of severe illness during the examination, but, on the contrary, fought vigorously with hands and feet against every touch, and kept up a continuous loud screaming until we had left the room.

Puffiness of the face was absent. Mouth and throat appeared perfectly normal. The excoriations at the nasal entrances were very marked, and were covered in part by crusts. The entire lower nose appeared red and swollen. This condition could possibly have been caused by mechanical and chemical irritation during the frequent spraying of the nose.

Besides these excoriations, the rhinitis, the high temperature and the report of slight albuminuria, I found multiple, hard and very small swollen lymph nodes along the posterior half of the neck, especially between the deeper muscles. In size the nodes varied between those of a pin's head and those of a small pea. They were not visible and could not be felt by superficial touch, but only by thorough palpation. This finding, together with the other symptoms, compelled me (in the discussion of the case) to diagnosticate a primary nasal diphtheria followed by secondary nephritis and retro-cervical adenitis, in spite of the negative bacteriological report. Accordingly, I suggested immediate serum treatment, which, even in case of a negative result, would have a far greater diagnostic value than a single culture. As the local colonization of diphtheric bacilli in the upper nose and the nasopharynx could not be very extensive (because by this time pseudomembranes would have been visible in the pharynx), and as the high fever and the

cervical lymph nodular infiltrations showed a marked systemic infection, I expected that Behring's serum brought into the blood of this child would develop a specially quick and energetic action in this case. All other local or systemic therapeutic measures were to be set aside.

Two thousand units of diphtheria antitoxin were injected the same evening, and on the following morning the father reported by telephone that the temperature of the child had dropped nearly to 99° F.

Not until the fourth culture was examined, eight days after this consultation, were Löffler bacilli found in the nasal secretion. The discharge from the ears persisted for two weeks, causing occasional variations in temperature. No search for Löffler bacilli was made in this secretion. I had only seen the patient once. The history of this case proves

(1) That a negative report as to the presence of Löffler bacilli is of *no value* in a doubtful case of diphtheria; and

(2) That a serum test is indicated *wherever* diphtheria is suspected.

DIPHTHERIA OF THE TONGUE, THE LIPS AND THE CONJUNCTIVAE WITH LITTLE SYSTEMIC INFECTION

While in the prevoius case the local diphtheria was hidden and of small dimensions and the systemic symptoms were severe, quite the reverse presents itself in the following.

M. G., male, age seven weeks, was brought to my clinic on October 3, 1902. Emaciated by gastroenteritis since birth, although fed by good mother's milk, the child weighed but eight pounds. During the last week frequent and copious sour smelling stools. Extensive intertrigo with numerous excoriations covered with a grayish smear, involving the buttocks and the scrotum. Rectal temperature 99° F. Appetite fair. Acid eructations. Restless at night. Adipose tissue absent. Skin light gray, forming folds. Lung, heart, liver, spleen and urine normal.

The eyelids are puffy and closed. Scant watery nasal discharge. The mucosa of both lips are covered by a white-grayish thick continuous pseudomembrane, extending from one corner of the mouth to the other. No fetor ex ore. No lymph nodular swelling about the neck.

A diagnosis, comprising gastroenteritis, intertrigo and stomatitis so far appeared correct but for the unusual extent and the thickness of the exudate on the lips and the entire absence of odor, which in particular excluded mercurial stomatitis, although calomel had been used during the last few days. The low temperature also appeared suspicious.

On turning the puffy eyelids the same white, continuous membrane presented itself on the upper and lower conjunctiva, which could easily be detached in toto. Pus was absent.

Inspection of the pharynx revealed pale and normal mucosa, but the entire anterior third of the tongue, above and below, was also covered with a continuous white pseudomembrane. The border of this exudate presented no infiltration and no edema.

The clinical diagnosis of diphtheria here had to rest on the conjunctival exudate and on the absence of odor from the mouth. As the pseudomembranes on the lips and on the tongue presented themselves before the eyelids were turned, it will appear but explicable that the absence of oral odor at first only seemed strange, for such an extensive diphtheria of both lips and the tongue (without throat involvement) so far I had not seen nor read of. The corresponding films upon the conjunctivæ naturally at once left no doubt as to the true nature of the case, for conjunctival diphtheria in infants is not infrequently met with. Diphtheria of the lips is rare, excepting where the whole oral cavity and the pharynx are involved. Diphtheria of the anterior third of the tongue without pharyngeal involvement and without severe systemic infection has not, to my knowledge, been described.

Two thousand units of diphtheria antitoxin were injected and, excepting the regulation of diet, rectal irrigations and frequent bathing of the parts covered by the intertrigo (then dried and dusted over by fullers' earth), no other therapeutic measures were used. After three days the pseudomembranes on the conjunctivæ and on the lips had disappeared entirely and those on the tongue to about one-third of their original size. This lingual exudate vanished after two more days. The excoriations on the buttocks and the scrotum also healed within a week. The general condition of the baby had been so improved within a few days, that the apparently contented child, looking up with clear eyes, could hardly be recognized.

Cultures taken from the surface of the pseudomembranes on the conjunctivæ, the lips, the tongue and the excoriations of the buttocks revealed but small numbers of Löffler bacilli (corresponding with the usual experience), while in the watery secretion of the nose (where no pseudomembranes were visible) diphtheria bacilli were found during the three following weeks. Cultures taken from the nipples of the maternal breast proved negative.

Astonishing in this case is the expansion of the local colonies of diphtheric organisms with so little systemic poisoning. A doubt as to whether the organisms found were true or pseudo-diphtheria bacilli cannot here be entertained, because Behring's serum proved effective. The question of virulence of the bacilli in this case can be left to a discussion among bacteriologists, for prior to the introduction of serumtherapy this patient would have beyond a doubt succumbed to this infection. That no postdiphtheric paralyses presented themselves after this manifold and extensive surface infection in so young a human organism, weakened by enteritis since birth, is another fact for speculation. This shows that not alone clinicians, but also bacteriologists, have as yet many unanswered questions to solve.

PARALYSIS OF THE SOFT PALATE FROM HIDDEN DIPHTHERIA

On April 8, 1903, I was requested to see the infant son of Mr. H. St. G., in Weehawken Heights, N. J., in consultation with his family physician.

The baby, eight weeks old, nursed by the mother, had appeared normal until six days ago, when fever up to 101.5° F. and apparent difficulty in deglutition had set in. Mild tonsillitis and pharyngitis were found by the family physician, without exudate, disappearing after three days, when the temperature returned to 97.5° F. But swallowing had become even more difficult, so that all efforts in that direction had ceased. Mother's milk poured into the mouth of the baby caused attacks of cough and of asphyxiation.

When seen by me on the evening of the seventh day of illness the child had not swallowed food for seventy-two hours. It was resting on its back, listless and languid. The pulse was weak and slow, the pupils dilated, the outer skin cool to the touch, the rectal temperature 96.5° F. The apathy of the child was striking.

No nasal discharge, the tongue was furred. The surface of the pharynx and of the tonsils appeared pale and normal. On palpation, multiple hard infiltration of the posterior cervical lymph nodes was found between the deeper muscles of the neck.

On close inspection of the throat it now became apparent that although attempts at vomiting were caused by the introduction of the tongue depressor, the muscles of the velum palati and of the uvula took no part in these contractions, and that instead these parts remained motionless even when touched by the spoon. This proved a paralysis of the soft palate, which fully explained the inability of the child to swallow.

The narrow space did not permit direct nasopharyngeal palpation.

In diagnosing this case the paralysis of the soft palate had to be primarily considered, because it presented the only definite symptom. The family physician, who did not notice the paralysis, had thought of a possible cerebral affection to which the marked apathy, the slow pulse and the dilated and slow-reacting pupils seemed to point.

In the discussion of this case I excluded a central cause for the pharyngeal paralysis for the following reasons:

(1) The paralysis appeared bilaterally well marked and equally divided on both sides. Tumor, cerebral abscess, hemorrhage and syphilitic endarteritis would each have caused unilateral symptoms.

(2) The regularity of the pulse and the absence of all convulsive symptoms excluded the possibility of meningitis.

(3) The apathy, the slow pulse, the cool skin, the flabbiness of the muscles and the subnormal temperature could all have been caused alone by starvation during the last three days, while again, these symptoms might be accepted as resulting in part at least from the general diphtheric infection accompanying the diphtheric pharyngeal paralysis.

Considered from the practical standpoint of the clinician, I was, in fact, forced to diagnosticate "early diphtheric pharyngeal paralysis" for the reason that in this manner alone the chances for a cure through Behring's serum were not diminished by delay. The multiple lymph nodular infiltration along the posterior neck presented plain evidence that absorption of infectious material from the nasopharynx was still continuing, and as the same infection had

most likely caused the palatal paralysis, no other diagnosis than that of diphtheria could be made. The very early onset of the paralysis was unusual, but for all we knew the diphtheria might have been present for days before the family physician was called, and, besides, the extreme youth of the patient might have been in part to blame for this.

In accord with this diagnosis 2,000 units of diphtheria antitoxin were injected immediately (I always carry this with me), the mother's breasts were pumped and the milk introduced into the stomach by means of a catheter and a funnel. No other therapy was resorted to.

Before departing, the fact was elicited from the family that an aunt of the baby (who had carried the child about more than the mother) had suffered from a sore throat during the previous week without medical attendance. An immediate inspection of this aunt's throat revealed the presence of a remaining strip of diphtheric pseudomembrane on the right tonsil.

Cultures taken the next day from the nose and the nasopharynx of the baby and examined in the bacteriological laboratory of the New York Health Department showed the presence of Löeffler bacilli in large numbers and in almost pure culture.

The injection of the antitoxin and the regular feeding by catheter improved the child's general condition within twenty-four hours. The paralysis remained stationary for another seven days, then slowly improved and disappeared entirely after twelve days.

The chief points of interest in this case are:

(1) The youth of the patient (eight weeks).

(2) The early appearance of diphtheric paralysis.

(3) The absence of visible diphtheria.

In conclusion, I would like to emphasize two points. The first is the diagnostic value of multiple retrocervical lymph nodular swelling in young children. If no outer source is visible, like eczema capillitii, this phenomenon usually points to nasopharyngeal infection. Acute catarrhal rhinitis is not associated with the symptom, but in chronic postnasal catarrh (so often met with even in very young children) it is always present, but here the infiltrated lymph nodes are usually larger and appear singly, scattered about the neck, probably because more infectious material could collect

during the longer time and because very likely only certain portions of the mucosa are affected.

In early diphtheric invasion of the nasopharynx I have found different conditions. No doubt owing to the greater extension of diphtheria (in comparison to catarrh), we here usually find many small and hard lymph nodes, unevenly distributed, but in astonishing numbers. This multiple acute swelling of lymph nodes in this locality is usually due to extensive surface infection of the nasopharynx. The most important possibility is diphtheria. If this lymph nodular infection is found in acute rhinitis of children, an injection of diphtheria antitoxin without delay is indicated, to my mind at least, and this even in cases where the temperature is normal or but slightly elevated.

The second point to be emphasized here is the doubtful value of an early bacteriological finding. Chance undoubtedly here usually decides the issue. To my mind the clinical diagnosis should guide our therapy in all acute infections. I need hardly refer to the tardy Widal test in typhoid. A negative bacteriological report in suspected diphtheria is absolutely worthless, while to wait for a positive report before injecting antitoxin may cause the period of ailment to pass in which this remedy could as yet have been curative. Exudate in a throat is highly suspicious of true diphtheria in every case, and here I rather prefer to rely on a serum test made in the body of the patient by an antitoxin injection than on a search for bacilli in a laboratory. The first means prompt action, effective treatment and safety to the patient; and if ineffective, at least the definite knowledge that diphtheria is absent. It has never occurred to me to treat the every-day cases of tonsillitis with antitoxin, but I do use it instantly where pseudomembranes form.

The bacteriological report later on need not interfere with the diagnosis previously made by inspection and proved or disproved by the positive or negative action of the serum on the patient.

As the serum treatment of diphtheria has been tested now for fully ten years, the time ought to have arrived when it is not looked upon as a "last resort" and given after waiting for days, but as the *only correct treatment* and the *safest test* to firmly establish the diagnosis, and therefore should be given on the *first day* of medical attendance.

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